WEST Search History

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DATE: Tuesday, October 10, 2006

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L35	L28 and (therapeutic)adj(effectiveness)	2
☐	L34	L28 and chylomicron	3
☐	L33	L28 and (VLDL)same(IDL)same(C)adj(reactive)adj(protein)	0
☐	L32	L28 and (hemostatic)adj(dysfunction)	0
☐	L31	L28 and (CRP)adj(complex)	0
☐	L30	L28 and (effectiveness)adj(of)adj(therapeutic)adj(agent)	0
☐	L29	L28 and (therapeutic)adj(testing)	0
☐	L28	436/507,515,516,517.ccls.	809
☐	L27	L24 and (hemostatic)adj(dysfunction)	0
☐	L26	L24 and (C)adj(reactive)adj(protein)	2
☐	L25	L24 and (C)adj(reactive)adj(protein)adj(complex)	0
☐	L24	435/11.ccls.	404
☐	L23	L22 and (CRP)	1
☐	L22	(walker)adj(john)adj(b)	13
☐	L21	(toh)adj(cheng)adj(hock)	9
☐	L20	(hock)adj(cheng)	0
☐	L19	L18 and (hemostatic)adj(dysfunction)	2
☐	L18	(tejidor)adj(liliana)	18
☐	L17	(tejidor)adj(liliana)adj(t)	0
☐	L16	(samis)adj(john)adj(a)	2
☐	L15	(nesheim)adj(mike)	2
☐	L14	(downey)adj(colin)	7
☐	L13	L11 and (C)adj(reactive)adj(protein)	0
☐	L12	L11 and CRP	0
☐	L11	(fisher)adj(timothy)adj(j)	8
☐	L10	L9 and (effectiveness)same(therapeutics)	4
☐	L9	L5 and (disseminated)adj(intravascular)adj(coagulation)	22
☐	L8	L5 and chylomicron	4
☐	L7	L5 and IDL	3
☐	L6	L5 and VLDL	6
☐	L5	L2 and (c)adj(reactive)adj(protein)same(complex)	60

DB=USPT; PLUR=YES; OP=OR

☐	L4	US-6429017-B1.did.	1

DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR

☐	L3	L2 and (hemostatic)adj(dysfunction)	4
☐	L2	prognosis	22084
☐	L1	(testing)same(effectiveness)same(hemostatic)adj(dysfunction)	0

END OF SEARCH HISTORY

Refine Search

Search Results -

Terms	Documents
L64 and diagnosis	13

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L66

Refine Search

Recall Text Clear Interrupt

Search History

DATE: Tuesday, October 10, 2006 **Purge Queries** Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L66	L64 and diagnosis	13	L66
L65	L64 and diagonisis	0	L65
L64	L63 and chylomicrons	17	L64
L63	L62 and IDL	25	L63
L62	L61 and VLDL	78	L62
L61	L60 and complex	807	L61
L60	L52 and (serum)adj(amyloid)adj(A)	868	L60
L59	L57 and inflammation	39	L59
L58	L57 and (hemostatic)adj(dysfunction)	1	L58
L57	L56 and chylomicrons	44	L57
L56	L55 and LDL	108	L56
L55	L54 and VLDL	119	L55
L54	L53 and complex	1314	L54
L53	L52 and (C)adj(reactive)adj(protein)	1573	L53

L52	436/69 71, 79, 507, 536.ccls.	2096900	L52
L51	L40 and (hemostatic)adj(dysfunction)	15	L51
L50	L46 and (hemostatic)adj(dysfunction)	1	L50
L49	L46 and chylomicrons	2	L49
L48	L46 and IDL	2	L48
L47	L46 and VLDL	4	L47
L46	L40 and (serum)adj(amyloid)adj(A)	53	L46
L45	L41 and chylomicrons	2	L45
L44	L41 and IDL	3	L44
L43	L42 and VLDL	7	L43
L42	L41 and lipoprotein	21	L42
L41	L40 and (c)adj(reactive)adj(protein)	68	L41
L40	(clotting)adj(assays)	1074	L40
DB=USPT; PLUR=YES; OP=OR			
L39	6429017.pn.	1	L39
L38	6321164.pn.	1	L38
L37	6101449.pn.	1	L37
L36	5708591.pn.	1	L36
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L35	L28 and (therapeutic)adj(effectiveness)	2	L35
L34	L28 and chylomicron	3	L34
L33	L28 and (VLDL)same(IDL)same(C)adj(reactive)adj(protein)	0	L33
L32	L28 and (hemostatic)adj(dysfunction)	0	L32
L31	L28 and (CRP)adj(complex)	0	L31
L30	L28 and (effectiveness)adj(of)adj(therapeutic)adj(agent)	0	L30
L29	L28 and (therapeutic)adj(testing)	0	L29
L28	436/507,515,516,517.ccls.	809	L28
L27	L24 and (hemostatic)adj(dysfunction)	0	L27
L26	L24 and (C)adj(reactive)adj(protein)	2	L26
L25	L24 and (C)adj(reactive)adj(protein)adj(complex)	0	L25
L24	435/11.ccls.	404	L24
L23	L22 and (CRP)	1	L23
L22	(walker)adj(john)adj(b)	13	L22
L21	(toh)adj(cheng)adj(hock)	9	L21
L20	(hock)adj(cheng)	0	L20
L19	L18 and (hemostatic)adj(dysfunction)	2	L19
L18	(tejidor)adj(liliana)	18	L18
L17	(tejidor)adj(liliana)adj(t)	0	L17
L16	(samis)adj(john)adj(a)	2	L16
L15	(nesheim)adj(mike)	2	L15
L14	(downey)adj(colin)	7	L14

L13	L11 and (C)adj(reactive)adj(protein)	0	L13
L12	L11 and CRP	0	L12
L11	(fisher)adj(timothy)adj(j)	8	L11
L10	L9 and (effectiveness)same(therapeutics)	4	L10
L9	L5 and (disseminated)adj(intravascular)adj(coagulation)	22	L9
L8	L5 and chylomicron	4	L8
L7	L5 and IDL	3	L7
L6	L5 and VLDL	6	L6
L5	L2 and (c)adj(reactive)adj(protein)same(complex)	60	L5
DB=USPT; PLUR=YES; OP=OR			
L4	US-6429017-B1.did.	1	L4
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L3	L2 and (hemostatic)adj(dysfunction)	4	L3
L2	prognosis	22084	L2
L1	(testing)same(effectiveness)same(hemostatic)adj(dysfunction)	0	L1

END OF SEARCH HISTORY

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NEWS 15	AUG 09	INSPEC enhanced with 1898-1968 archive
NEWS 16	AUG 28	ADISCTI Reloaded and Enhanced
NEWS 17	AUG 30	CA(SM)/CAplus(SM) Austrian patent law changes
NEWS 18	SEP 11	CA/CAplus enhanced with more pre-1907 records
NEWS 19	SEP 21	CA/CAplus fields enhanced with simultaneous left and right truncation
NEWS 20	SEP 25	CA(SM)/CAplus(SM) display of CA Lexicon enhanced
NEWS 21	SEP 25	CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS 22	SEP 25	CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS 23	SEP 28	CEABA-VTB classification code fields reloaded with new classification scheme

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=> s hemostatic dysfunction
L1          102 HEMOSTATIC DYSFUNCTION

=> s l1 and monitoring
L2            3 L1 AND MONITORING

=> dup remove l2
PROCESSING COMPLETED FOR L2
L3            3 DUP REMOVE L2 (0 DUPLICATES REMOVED)

=> d l3 1-3 cbib abs

L3   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
2006:538913   Document No. 145:23858  Method for diagnosing critically ill
     patients by measuring the formation of lipoprotein-C-reactive protein
     complex in the presence of a surfactant.  Jones, Gregory Ray;
     Borzhemskaya, Larisa; Hanson, Donald G.; Estevez, Rafael Angel; Wilson,
     Mark S.; Link, John Glenn; Barnes, Bryan (Biomerieux, Inc., USA).  PCT
     Int. Appl. WO 2006060386 A1 20060608, 37 pp.  DESIGNATED STATES: W:  AE,
     AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR,
     CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU,
     ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU,
     LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH,
     PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ,
     UA, UG, US, UZ, VC, VN, YU, ZA; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM,
     CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL,
     PT, SE, SN, TD, TG, TR.  (English).  CODEN: PIXXD2.  APPLICATION: WO
     2005-US43120 20051130.  PRIORITY: US 2004-632431P 20041201.
AB   Provided is an improved method of diagnosing and monitoring
     hemostatic dysfunction, sepsis-related morbidity or
     severe infection by improving detection of an in vitro complex formed by
     lipoprotein and C-reactive protein with the utilization of an effective
     amount of a surface active agent in the reagent.  The method includes: (a)
     obtaining a patient sample; (b) combining said sample with a reagent
     comprising a divalent cation and an effective amount of a surface active
     agent to form a reaction mixture; and (c) examining said reaction mixture to
determine
     whether an LC-CRP complex is formed to diagnose or monitor patients having
     hemostatic dysfunction, sepsis-related morbidity or
     severe infection.

L3   ANSWER 2 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
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2003:697159 Document No. 139:194022 Method for diagnosing and monitoring hemostatic dysfunction, severe infection and systematic inflammatory response syndrome. Toh, Cheng Hock; Tejidor, Liliana; Neisheim, Mike; Jones, Gregory (Biomerieux, Inc., USA). PCT Int. Appl. WO 2003073099 A1 20030904, 62 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US5980 20030227. PRIORITY: US 2002-359932P 20020227; US 2002-363073P 20020311; US 2002-396392P 20020717; US 2002-404652P 20020820.

AB A method for diagnosing and monitoring subjects for hemostatic dysfunction, severe infection and systematic inflammatory response syndrome is provided whereby lipoproteins are examined for abnormalities, particularly for prothrombinase enhancement, through quant. and qual. anal.

L3 ANSWER 3 OF 3 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

2001:305605 Document No.: PREV200100305605. The value of the APTT waveform in predicting mortality and haemostatic dysfunction. Toh, Cheng-Hock [Reprint author]; Downey, Colin [Reprint author]; Wenstone, Richard [Reprint author]; Paton, Ray [Reprint author]; Ticknor, Larry. Haematology, Anaesthesia and Computer Science, University of Liverpool, Liverpool, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 51a. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB The molecular mechanism underlying the biphasic APTT waveform, which is detected in patients with haemostatic dysfunction, has now been unravelled. This is due to a complex between very low or intermediate density lipoprotein and C-reactive protein (CRP) that is induced by calcium ions. An immediate, progressive drop from 100% light transmittance occurs and the degree of this change at 18 seconds (TL18) can be quantified on the MDA-180 automated haemostasis analyser. The clinical ramifications of lipoprotein complexed-CRP formation have now been further examined in the intensive care unit (ICU) setting. Over a 24-month period, 1187 consecutive patients admitted into the ICU were monitored through daily APTT waveform analysis. On admission, 242 patients exhibited a biphasic waveform with 370 others developing biphasic changes subsequently. Biphasic waveforms on admission were associated with the positive predictive value (PPV) for death of 52% as compared with 31% for all unscreened admissions. When the most severe change in the biphasic slope was correlated with outcome by selecting the lowest TL18 in any one patient, the association was even more striking. Data analysed by non-linear regression, showed a stepwise increase in mortality. The PPV for DIC also increased similarly although deaths in the 90 to 100% TL18 ranges were not predicted through the presence of overt DIC (Japanese MHW 1988 score). However, all deaths in this group had evidence of haemostatic dysfunction with elevated markers of thrombin generation. Conclusion: The APTT waveform can identify ICU patients at risk of an adverse outcome both on and during admission. It has the potential too of shedding light on the pathophysiological interactions between coagulation, inflammation and the lipid response.

=> s monitoring complex formation
L4 40 MONITORING COMPLEX FORMATION

=> s l4 and C reactive protein
L5 0 L4 AND C REACTIVE PROTEIN

=> s l4 and VLDL
L6 0 L4 AND VLDL

=> dup remove l4
PROCESSING COMPLETED FOR L4
L7 12 DUP REMOVE L4 (28 DUPLICATES REMOVED)

=> d l7 1-12 cbib abs

L7 ANSWER 1 OF 12 MEDLINE on STN DUPLICATE 1
2005109948. PubMed ID: 15741074. Monitoring complex formation in the blood-coagulation cascade using aptamer-coated SAW sensors. Gronewold T M A; Glass S; Quandt E; Famulok M. (Center of Advanced European Studies and Research, Aptamer Biosensors, Ludwig-Erhard-Allee 2, 53175 Bonn, Germany.) Biosensors & bioelectronics, (2005 Apr 15) Vol. 20, No. 10, pp. 2044-52. Journal code: 9001289. ISSN: 0956-5663. Pub. country: England: United Kingdom. Language: English.

AB Specific binding of the anticoagulants heparin and antithrombin III to the blood clotting cascade factor human thrombin was recorded as a function of time with a Love-wave biosensor array consisting of five sensor elements. Two of the sensor elements were used as references. Three sensor elements were coated with RNA or DNA aptamers for specific binding of human thrombin. The affinity between the aptamers and thrombin, measured using the biosensor, was within the same range as the value of K(D) measured by filter binding experiments. Consecutive binding of the thrombin inhibitors heparin, antithrombin III or the heparin-antithrombin III complex to the immobilized thrombin molecules, and binding of a ternary complex of heparin, anithrombin III, and thrombin to aptamers was evaluated. The experiments showed attenuation of binding to thrombin due to heparin-antithrombin III complex formation. Binding of heparin activated the formation of the inhibitory complex of antithrombin III with thrombin about 2.7-fold. Binding of the DNA aptamer to exosite II appeared to inhibit heparin binding to exosite I.

L7 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
2003:972257 Document No. 140:13701 Detection of mutation using bipartite capture probe immobilized on high density arrays. Van Beuningen, Marinus Gerardus Johannus (Pamgene B.V., Neth.). PCT Int. Appl. WO 2003102233 A1 20031211, 30 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-EP5749 20030602. PRIORITY: EP 2002-447108 20020603.

AB The present invention relates to methods for identifying analytes in a sample comprising the steps of: (a) incubating said analytes with a plurality of bipartite capture probes, said capture probes being immobilized in predefined regions on a solid substrate, and each capture probe consisting essentially of a first fragment which is at one end immobilized to said substrate and at the other end is complementary linked to a second fragment, wherein said second fragment comprises an extension fragment capable of identifying an analyte; (b) monitoring complex formation between sample analytes and extension fragments; (c) sequentially modifying complex formation conditions; allowing the release of captured analyte mols. from the substrate; and (d) detecting and identifying the released analytes. The present invention also relates to different uses of said methods as well as microarrays and kits for performing said methods. The bipartite capture probes are immobilized on microarray via photo labile, acid labile, base labile, enzyme labile, and oxidation labile linker. The captured analytes are released from substrate by temperature variation, base treatment, acid

treatment, oxidative treatment, enzymic treatment, and photolysis, including any sequentially combination thereof.

L7 ANSWER 3 OF 12 MEDLINE on STN DUPLICATE 2

2002165671. PubMed ID: 11796716. Plasminogen activator inhibitor type 1 promotes the self-association of vitronectin into complexes exhibiting altered incorporation into the extracellular matrix. Minor Kenneth H; Peterson Cynthia B. (Department of Biochemistry and Cellular and Molecular Biology, University of Tennessee, Knoxville, Tennessee 37996, USA.) The Journal of biological chemistry, (2002 Mar 22) Vol. 277, No. 12, pp. 10337-45. Electronic Publication: 2002-01-16. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB Serine proteinase inhibitors, including plasminogen activator inhibitor type 1 (PAI-1) and antithrombin, are key regulators of hemostatic processes such as thrombosis and wound healing. Much evidence suggests that PAI-1 can influence such processes, as well as pathological events like tumor metastasis, through its ability to directly regulate binding of blood platelets and cells to extracellular substrata. One way that PAI-1 influences these processes may be mediated through its binding to the plasma protein vitronectin. Binding to PAI-1 results in the incorporation of vitronectin into a higher order complex with a potential for multivalent interactions (Podor, T. J., Shaughnessy, S. G., Blackburn, M. N., and Peterson, C. B. (2000) J. Biol. Chemical 275, 25402-25410). In this study, evidence is provided to support this concept from studies on the effects of PAI-1-induced multimerization on the interactions of vitronectin with matrix components and cell surface receptors. By monitoring complex formation and stability over time using size-exclusion high performance liquid chromatography, a correlation is made between PAI-1-induced multimerization and enhanced cell/matrix binding properties of vitronectin. This evidence indicates that PAI-1 alters the adhesive functions of vitronectin by converting the protein via the higher order complex to a self-associated, multivalent species that is functionally distinct from the abundant monomeric form found in the circulation.

L7 ANSWER 4 OF 12 MEDLINE on STN DUPLICATE 3

2000193616. PubMed ID: 10727931. Mouse Hsp25, a small shock protein. The role of its C-terminal extension in oligomerization and chaperone action. Lindner R A; Carver J A; Ehrnsperger M; Buchner J; Esposito G; Behlke J; Lutsch G; Kotlyarov A; Gaestel M. (Department of Chemistry, University of Wollongong, Australia.) European journal of biochemistry / FEBS, (2000 Apr) Vol. 267, No. 7, pp. 1923-32. Journal code: 0107600. ISSN: 0014-2956. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB Under conditions of cellular stress, small heat shock proteins (sHsps), e.g. Hsp25, stabilize unfolding proteins and prevent their precipitation from solution. 1H NMR spectroscopy has shown that mammalian sHsps possess short, polar and highly flexible C-terminal extensions. A mutant of mouse Hsp25 without this extension has been constructed. CD spectroscopy reveals some differences in secondary and tertiary structure between this mutant and the wild-type protein but analytical ultracentrifugation and electron microscopy show that the proteins have very similar oligomeric masses and quaternary structures. The mutant shows chaperone ability comparable to that of wild-type Hsp25 in a thermal aggregation assay using citrate synthase, but does not stabilize alpha-lactalbumin against precipitation following reduction with dithiothreitol. The accessible hydrophobic surface of the mutant protein is less than that of the wild-type protein and the mutant is also less stable at elevated temperature. 1H NMR spectroscopy reveals that deletion of the C-terminal extension of Hsp25 leads to induction of extra C-terminal flexibility in the molecule. Monitoring complex formation between Hsp25 and dithiothreitol-reduced alpha-lactalbumin by 1H NMR spectroscopy indicates that the C-terminal extension of Hsp25 retains its flexibility during this interaction. Overall, these data suggest that a highly flexible C-terminal extension in mammalian sHsps is required for

full chaperone activity.

L7 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
2000:787891 Document No. 134:71800 Low affinity carbohydrate lectin interactions examined with surface plasmon resonance. Weimar, Thomas; Haase, Bernd; Kohli, Thies (Institut fur Chemie, Medizinische Universitat zu Lubeck, Lubeck, D-23538, Germany). Journal of Carbohydrate Chemistry, 19(8), 1083-1089 (English) 2000. CODEN: JCACDM. ISSN: 0732-8303. Publisher: Marcel Dekker, Inc..
AB Two lectins where the structures of bound carbohydrates are being studied in our laboratory by NMR spectroscopy are the lectins Viscum album agglutinin I (VAA I) and Aleuria aurantia agglutinin (AAA). Transferred NOE (trNOE) expts., from which the structures of the bound carbohydrates can be deduced, have been reported for the complexes of VAA I and β-D-Gal-(1→2)-3-D-Gal-(1→O)-Me and AAA and α-L-Fuc-(1→6)-3-D-GIcNAc-(1→O)-Me. Surface plasmon resonance (SPR) technol. appeared to be an attractive method to achieve this goal since it can be used to study the interactions between biomols. by monitoring complex formation through the increase of mass of a surface where one mol. is immobilized and a binding partner is present in the mobile phase.

L7 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
2000:328725 Equilibrium studies of ethidium bromide binding to synthetic polynucleotides.. Johnston, Danielle M.; Fisher, Matthew A. (Department of Chemistry, Saint Vincent College, Latrobe, PA, 15650, USA). Book of Abstracts, 219th ACS National Meeting, San Francisco, CA, March 26-30, 2000, CHED-584. American Chemical Society: Washington, D. C. (English) 2000. CODEN: 69CLAC.
AB The equilibrium binding of ethidium bromide to synthetic polynucleotides was examined to better understand sequence preferences in ethidium binding. We used the increased fluorescence of the complex as compared to free ethidium as a means of monitoring complex formation. Previous expts. using dinucleotides determined that ethidium prefers binding to GC base pairs within DNA because of decreased stacking consts. We examined the binding of ethidium to various synthetic polynucleotides to determine if this sequence preference still existed in larger mols. Preliminary results show that the binding consts. for the polynucleotides are lower than that of natural DNA and similar for both AT and GC polymers. However, the intensity constant for bound ethidium varies with polynucleotide composition The possible role of the rigidity of the synthetic polymer will be examined

L7 ANSWER 7 OF 12 MEDLINE on STN DUPLICATE 4
1998175922. PubMed ID: 9506960. Control of the DnaK chaperone cycle by substoichiometric concentrations of the co-chaperones DnaJ and GrpE. Pierpaoli E V; Sandmeier E; Schonfeld H J; Christen P. (Biochemisches Institut, Universitat Zurich, Winterthurerstr. 190, CH-8057 Zurich, Switzerland.) The Journal of biological chemistry, (1998 Mar 20) Vol. 273, No. 12, pp. 6643-9. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.
AB The polypeptide binding and release cycle of the molecular chaperone DnaK (Hsp70) of Escherichia coli is regulated by the two co-chaperones DnaJ and GrpE. Here, we show that the DnaJ-triggered conversion of DnaK.ATP (T state) to DnaK.ADP.Pi (R state), as monitored by intrinsic protein fluorescence, is monophasic and occurs simultaneously with ATP hydrolysis. This is in contrast with the T-->R conversion in the absence of DnaJ which is biphasic, the first phase occurring simultaneously with the hydrolysis of ATP (Theyssen, H., Schuster, H.-P., Packschies, L., Bukau, B., and Reinstein, J. (1996) J. Mol. Biol. 263, 657-670). Apparently, DnaJ not only stimulates ATP hydrolysis but also couples it with conformational changes of DnaK. In the absence of GrpE, DnaJ forms a tight ternary complex with peptide.DnaK.ADP.Pi (Kd = 0.14 microM). However, by monitoring complex formation between DnaK (1 microM) and a fluorophore-labeled peptide in the presence of ATP (1 mM),

DnaJ (1 microM), and varying concentrations of the ADP/ATP exchange factor GrpE (0.1-3 microM), substoichiometric concentrations of GrpE were found to shift the equilibrium from the slowly binding and releasing, high-affinity R state of DnaK completely to the fast binding and releasing, low-affinity T state and thus to prevent the formation of a long lived ternary DnaJ. substrate.DnaK.ADP.Pi complex. Under in vivo conditions with an estimated chaperone ratio of DnaK:DnaJ:GrpE = 10:1:3, both DnaJ and GrpE appear to control the chaperone cycle by transient interactions with DnaK.

L7 ANSWER 8 OF 12 MEDLINE on STN DUPLICATE 5

97320125. PubMed ID: 9177009. Study of haptoglobin-hemoglobin complexes by titration curves, capillary electrophoresis and capillary isoelectric focusing. Righetti P G; Conti M; Gelfi C. (Department of Agricultural and Industrial Biotechnology, University of Verona, Italy.) Journal of chromatography. A, (1997 Apr 11) Vol. 767, No. 1-2, pp. 255-62. Journal code: 9318488. ISSN: 0021-9673. Pub. country: Netherlands. Language: English.

AB A novel method is described for monitoring complex formation between macromolecules, based on combined isoelectric focusing-electrophoresis in capillaries. The example studied is the binding of serum haptoglobin (Hp) to hemoglobin (Hb). A known amount of Hb is focused in a capillary in a pH 6-8 range (pI of Hb = 7.0) and thus kept temporarily "immobilized" in the electrophoretic chamber. Subsequently, increasing amounts of ligand (Hp) are loaded cathodically and allowed to sweep past the focused Hb zone. As the complex formed has a pI value well-outside the bounds of such a pH gradient (the 1:1 molar Hb-Hp complex has a pI of 5.5, the 1 to 1/2 molar Hp-Hb complex has a pI of 5.0) it escapes immobilization and moves past the detector window, where it is monitored and quantified. Since the detector is set at 416 nm, where only Hb absorbs, and since the molar extinction coefficient of Hb is well known, it is quite easy to calculate the molar amount of Hb bound to the complex. As an additional check, the amount of unreacted Hb can now be mobilized by disrupting the pH gradient and allowing this residual free Hb to also reach the detector and be quantified. The method is easy, fast, simple and fully automated and thus could represent a valid alternative to existing methods in clinical chemistry for quantifying the amount of Hp in human sera in pathological conditions, such as hemolytic anemias and transfusion reactions.

L7 ANSWER 9 OF 12 MEDLINE on STN DUPLICATE 6

97160482. PubMed ID: 9007982. Recombinant hirustasin: production in yeast, crystallization, and interaction with serine proteases. Di Marco S; Fendrich G; Knecht R; Strauss A; Pohlig G; Heim J; Priestle J P; Sommerhoff C P; Grutter M G. (Core Drug Discovery Technologies, Pharmaceuticals Division, Ciba-Geigy Limited, Basel, Switzerland.. dimarco@fmi.ch) . Protein science : a publication of the Protein Society, (1997 Jan) Vol. 6, No. 1, pp. 109-18. Journal code: 9211750. ISSN: 0961-8368. Pub. country: United States. Language: English.

AB A synthetic gene coding for the 55-amino acid protein hirustasin, a novel tissue kallikrein inhibitor from the leech Hirudo medicinalis, was generated by polymerase chain reaction using overlapping oligonucleotides, fused to the yeast alpha-factor leader sequence and expressed in Saccharomyces cerevisiae. Recombinant hirustasin was secreted mainly as incompletely processed fusion protein, but could be processed in vitro using a soluble variant of the yeast yscF protease. The processed hirustasin was purified to better than 97% purity. N-terminal sequence analysis and electrospray ionization mass spectrometry confirmed a correctly processed N-terminus and the expected amino acid sequence and molecular mass. The biological activity of recombinant hirustasin was identical to that of the authentic leech protein. Crystallized hirustasin alone and in complex with tissue kallikrein diffracted beyond 1.4 A and 2.4 A, respectively. In order to define the reactive site of the inhibitor, the interaction of hirustasin with kallikrein, chymotrypsin, and trypsin was investigated by monitoring complex

formation in solution as well as proteolytic cleavage of the inhibitor. During incubation with high, nearly equimolar concentration of tissue kallikrein, hirustasin was cleaved mainly at the peptide bond between Arg 30 and Ile 31, the putative reactive site, to yield a modified inhibitor. In the corresponding complex with chymotrypsin, mainly uncleaved hirustasin was found and cleaved hirustasin species accumulated only slowly. Incubation with trypsin led to several proteolytic cleavages in hirustasin with the primary scissile peptide bond located between Arg 30 and Ile 31. Hirustasin appears to fall into the class of protease inhibitors displaying temporary inhibition.

L7 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
1995:538908 Document No. 122:287502 Factor Xa-Factor Va Complex Assembles in Two Dimensions with Unexpectedly High Affinity: An Experimental and Theoretical Approach. Ye, Jia; Esmon, Charles T. (Health Sciences Center, University of Oklahoma, Oklahoma City, OK, 73104, USA). Biochemistry, 34(19), 6448-53 (English) 1995. CODEN: BICHAW. ISSN: 0006-2960. Publisher: American Chemical Society.
AB The influence of phospholipid vesicle concentration and size on the affinity and
the kinetics of assembly of the prothrombin activation complex are examined Activation of prethrombin 1 was used to monitor complex formation between factors Va and Xa. When activation rates were measured immediately after the addition of the reactants, the rate of activation increased, and subsequently decreased, as a function of increasing vesicle concentration Larger
vesicles did not inhibit the reaction to a comparable extent until much higher phospholipid concns. were present. The inhibition by high vesicle concns. was significantly reduced by a prolonged incubation period. These results are interpreted as an initial step of factors Va and Xa binding independently to sep. phospholipid vesicles, followed by a slow redistribution between vesicles to maximize complex formation. These expts. indicated that the Kd ≤25 pM, much tighter than previously reported. Two-dimensional binding on the membrane surface was investigated under conditions where all of the proteins were membrane bound. The complex formation was independent of the surface d. of the reactants, indicating a near complete complex formation at the lowest surface d. of the reactants. Thus, we conclude that (i) the overall affinity of factor Va-factor Xa interaction in the presence of vesicles is higher than previously appreciated, and (ii) factor Va and factor Xa complex once they bind to the same vesicle.

L7 ANSWER 11 OF 12 MEDLINE on STN DUPLICATE 7
96001348. PubMed ID: 7548163. Production and characterization of recombinant human proteinase inhibitor 6 expressed in Pichia pastoris. Sun J; Coughlin P; Salem H H; Bird P. (Department of Medicine, Monash Medical School, Box Hill, Australia.) Biochimica et biophysica acta, (1995 Sep 27) Vol. 1252, No. 1, pp. 28-34. Journal code: 0217513. ISSN: 0006-3002. Pub. country: Netherlands. Language: English.
AB The human intracellular serine proteinase inhibitor, proteinase inhibitor 6 (PI-6), was expressed in the methylotropic yeast Pichia pastoris. The PI-6 cDNA was modified to encode six histidine residues immediately after the initiation codon, and was placed under the control of the P. pastoris alcohol oxidase promoter in the vector pHIL-D2. On the methanol induction, active recombinant PI-6 was produced within the yeast cells, and following cell lysis, was separated from yeast proteins by affinity chromatography using nickel nitrilo-tri-acetic acid (NTA) resin. The interaction of recombinant PI-6 with a range of serine proteinases was studied. Second order association rate constants (ka) were derived for the interaction with trypsin (1.8 x 10(6) M-1 s-1), thrombin (1.2 x 10(5) M-1 s-1), urokinase plasminogen activator (4.0 x 10(4) M-1 s-1), plasmin (1.3 x 10(6) M-1 s-1), and activated protein C (7.5 x 10(3) M-1 s-1). By monitoring complex formation, recombinant PI-6
was also shown to interact with factor Xa. No complex formation was observed with chymotrypsin, human leukocyte elastase, cathepsin G and

tissue plasminogen activator, although PI-6 is apparently a substrate for chymotrypsin, leukocyte elastase and cathepsin G.

L7 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
1993:594486 Document No. 119:194486 Dynamic molybdenum-95 NMR studies of a series of molybdenum-copper-sulfur complexes containing R2dtc (dithiocarbamate) ligands. Cao, Rong; Lei, Xinjin; Xie, Xiulan; Huang, Zhiying; Hong, Maochun; Liu, Hanqin (Fuzhou Lab. Struct. Chem., Fuzhou, Peop. Rep. China). Fenxi Ceshi Tongbao, 11(5), 40-2 (Chinese) 1992. CODEN: FCTOE8. ISSN: 1000-3800.
AB Dynamic 95Mo NMR was used to monitor the reactions in the CuCl/NaS2CNR2/MoOnS4-n2- (n = 0, 2) mixts. (molar ratio 3:3:1) in DMF and find the occurrences of different products and appropriate conditions. Compds. with MoxCuy (x = 1, 2; yr = 1, 2, 3, 4, 5) cores were found.

=> d his

(FILE 'HOME' ENTERED AT 10:35:07 ON 10 OCT 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 10:35:34 ON 10 OCT 2006
L1 102 S HEMOSTATIC DYSFUNCTION
L2 3 S L1 AND MONITORING
L3 3 DUP REMOVE L2 (0 DUPLICATES REMOVED)
L4 40 S MONITORING COMPLEX FORMATION
L5 0 S L4 AND C REACTIVE PROTEIN
L6 0 S L4 AND VLDL
L7 12 DUP REMOVE L4 (28 DUPLICATES REMOVED)

=> s l1 and complex formation
L8 0 L1 AND COMPLEX FORMATION

=> s complex formation
L9 178209 COMPLEX FORMATION

=> s l9 and hemostatic dysfunction
L10 0 L9 AND HEMOSTATIC DYSFUNCTION

=> s l9 and CRP
L11 271 L9 AND CRP

=> s l11 and VLDL
L12 6 L11 AND VLDL

=> dup remove l12
PROCESSING COMPLETED FOR L12
L13 6 DUP REMOVE L12 (0 DUPLICATES REMOVED)

=> d l13 1-6 cbib abs

L13 ANSWER 1 OF 6 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
2004141454 EMBASE Prothrombinase enhancement through quantitative and qualitative changes affecting very low density lipoprotein in complex with C-reactive protein. Dennis M.W.; Downey C.; Brufatto N.; Nesheim M.E.; Stevenson K.; Toh C.H.. Dr. C.H. Toh, Roald Dahl Haemostasis/Thromb. Ctr., Royal Liverpool University Hospital, Prescot Street, Liverpool L7 8XP, United Kingdom. toh@liverpool.ac.uk. Thrombosis and Haemostasis Vol. 91, No. 3, pp. 522-530 2004.
Refs: 35.
ISSN: 0340-6245. CODEN: THHADQ
Pub. Country: Germany. Language: English. Summary Language: English.
Entered STN: 20040412. Last Updated on STN: 20040412
AB The biphasic waveform that can predict for disseminated intravascular

coagulation (DIC) is due to the formation of a calcium-dependent complex between C reactive protein (CRP) and very low density lipoprotein (VLDL). As thrombin generation is pivotal to DIC, this aspect has been specifically investigated and the VLDL component has been found to increase prothrombinase activity via both quantitative and qualitative changes. The specific prothrombinase activity of VLDL from patients manifesting the biphasic waveform was 2.5 times that of normal individuals or critically ill patients without the biphasic waveform. This activity was due to an increase in anionic phospholipid surfaces that could be inhibited with excess annexin V and which was dependent on structurally intact apolipoprotein B. The qualitative change appeared to be due to a deficiency of phosphatidylethanolamine inVLDL from patients with the biphasic waveform.The functional consequence of this enhanced prothrombinase activity was an increased procoagulant effect in plasma. Using a modified activated partial thromboplastin time assay, the mean normal clot time decreased significantly when VLDL from patients with biphasic waveforms was substituted. These results indicate that VLDL derived from patients with the biphasic waveform can enhance thrombin procoagulant activity. As the CRP-VLDL complex exists in vivo, it could have a pathogenic role in disseminating the process of intravascular coagulation. .COPYRGT. 2004 Schattauer GmbH, Stuttgart.

L13 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2004:167455 Document No.: PREV200400161711. Procoagulant enhancement through quantitative and qualitative changes affecting very low density lipoprotein in complex with C-reactive protein. Toh, Cheng Hock [Reprint Author]; Dennis, Michael W. [Reprint Author]; Colin, Downey [Reprint Author]. Roald Dahl Haemostasis and Thrombosis Unit, Royal Liverpool University Hospital, Liverpool, UK. Blood, (November 16 2003) Vol. 102, No. 11, pp. 90b. print.
Meeting Info.: 45th Annual Meeting of the American Society of Hematology. San Diego, CA, USA. December 06-09, 2003. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB A complex between C reactive protein (CRP) and very low density lipoprotein (VLDL) is the molecular mechanism underlying the biphasic waveform that can predict for disseminated intravascular coagulation (DIG). To ascertain if it plays a role in the disease process rather than just as a predictive marker, we have established that there is a virtual 100% correlation to clinical DIC at maximal levels of complex formation in patient plasma. It also exists in vivo as demonstrated by size-exclusion chromatography and the quantitation of CRP within isolated VLDL fractions. We have now found that VLDL from patients with the biphasic waveform only supports increased prothrombinase activity via both quantitative VLDL triglyceride increases (up to 4 mM triglyceride) and qualitative changes with 2.5 times the specific prothrombinase activity of VLDL from normal or critically ill individuals without the biphasic waveform. This activity can be inhibited with excess annexin V at 5 mM CaCl2 and depends on intact apolipoprotein (apo) B but not apo E. Flow cytometric analysis after apo B immunoadsorption shows no intact VLDL particles suggesting that the role of apo B is as structural support for the requisite phospholipid conformation. Further qualitative assessment of the VLDL by 2 dimensional thin layer chromatography demonstrated an absence of phosphatidylethanolamine in VLDL from patients with the biphasic waveform only. To ascertain if these changes responsible for increasing thombin generation had functional consequences on coagulation in plasma, a modified activated partial thromboplastin time assay was used. The addition into normal plasma of isolated VLDL from patients with the biphasic waveform significantly shortened clot time. The mean clot time was 203 seconds (SEM 0.95) for VLDL from 7 normal unrelated donors and this decreased to 178 seconds (SEM 3.02) for VLDL from 7 patients with biphasic waveforms. There is dose-dependence of this effect as increasing proportional ratios of VLDL from patients admixed to

normal VLDL shortened the clot time proportionately in all 6 cases with a plateauing of effect in 2 experiments. Taken together, these results indicate that the CRP-VLDL complex can enhance the pro-coagulant aspects of thrombin generation. It could therefore have a pathogenic role in disseminating the process of intravascular coagulation.

L13 ANSWER 3 OF 6 MEDLINE on STN

2003382682. PubMed ID: 12918783. Lipoprotein-complexed C-reactive protein and the biphasic transmittance waveform in critically ill patients. Nesheim Michael; Samis John; Walker John; Becker Lev; Brufatto Nicole; Fischer Timothy; Tejidor Liliana; Jones Greg; Houdijk Wim; Giles Alan; Koschinsky Marlys; Wenstone Richard; Downey Colin; Toh Cheng Hock. (Department of Biochemistry, Queen's University, Kingston, Ontario, Canada.. nesheimm@post.queensu.ca) . Blood reviews, (2002 Dec) Vol. 16 Suppl 1, pp. S15-22. Journal code: 8708558. ISSN: 0268-960X. Pub. country: Scotland: United Kingdom. Language: English.

AB The 'biphasic transmittance waveform' (BTW) refers to a decrease in light transmittance that often occurs prior to clotting in coagulation assays of critically ill patient plasmas. It correlates with disseminated intravascular coagulation and mortality. The present work shows that the BTW is due to the rapid formation of a precipitate and a coincident change in turbidity in re-calcified plasma. The precipitate was isolated from patient plasma and contained lipids typical of very low density lipoprotein (VLDL), plus the proteins apolipoprotein B-100 and C-reactive protein (CRP). Precipitation also occurred in normal plasma supplemented with CRP. In addition, CRP precipitated with VLDL and intermediate density lipoprotein, but not low density lipoprotein or high density lipoprotein. The Kd value for the CRP/VLDL interaction is 340 nM. The IC50 value of Ca2+ for complex formation is 5.0 mM, and epsilon-aminocaproic acid inhibits the process. In 15 plasmas with the BTW from critically ill patients, CRP was highly elevated (77-398 microg/mL) and VLDL cholesterol ranged from 0.082 to 1.32 mM. The magnitude of the turbidity change on re-calcification correlated well with the calculated level of the CRP/ VLDL complex. Thus, the Ca2+-dependent formation of a complex between CRP and VLDL accounts for the BTW.

L13 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

2001:301143 Document No.: PREV200100301143. In vitro binding of C-reactive protein to very low and intermediate density lipoproteins in a calcium-dependent complex. Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L. [Reprint author]. Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB The presence of a biphasic aPTT waveform has been correlated to negative outcome for patients in the intensive care setting (Downey C, et al. Early Identification and Prognostic Implications in Disseminated Intravascular Coagulation through Transmittance Waveform Analysis. Thromb Haemost 1998; 80: 65-9). It has recently been discovered that the mechanism underlying the atypical biphasic aPTT waveform is the result of the formation of a complex between C-reactive protein (CRP) with very low and intermediate density lipoprotein (VLDL or IDL) in a divalent cation-dependent reaction. The complex has been termed LC-CRP for "Lipoprotein-Complexed C-Reactive Protein". Initially, it was demonstrated that CRP isolated from patient plasma formed the complex when added to normal plasma. A possible mechanism for complex formation could be the result of qualitative differences in either CRP or the lipoproteins. To address this,

recombinant CRP (rCRP) was combined in vitro with various lipoprotein subfractions. Free and bound CRP were quantified by ELISA. Results showed that rCRP was indistinguishable from patient CRP and that complex formation was specific for the VLDL/IDL subfraction. Recombinant CRP did not form a complex with LDL or HDL. Binding was inhibited by 1 mM phosphorylcholine, suggesting the involvement of the phosphatidylcholine lipid headgroups during complex formation. While these results may suggest that qualitative differences in CRP do not account for LC-CRP formation, they also enable the development of a quantitative assay for LC-CRP. An LC-CRP calibrator was constructed by mixing VLDL (0.24 mg/mL cholesterol) and CRP (200 mug/mL) in lipoprotein deficient plasma. By measuring the initial absorbance rate, dilutions in a plasma base provided a linear dose-response curve. As a synthetic VLDL substitute, artificial vesicles prepared from mixtures of phosphatidylcholine and lysophosphatidylcholine bound moderately to CRP. Development of a quantitative assay for LC-CRP that is fully automated and adaptable to multiple instrument platforms would allow direct measurement of the complex and has the potential to aid in therapeutic intervention and monitoring in this critically ill patient population.

L13 ANSWER 5 OF 6 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

93043144 EMBASE Document No.: 1993043144. Role of human C-reactive protein (CRP) in lipid metabolism: Ingestion of CRP-lipoproteins complex by macrophage. Kitao H.. Department of Laboratory Medicine, Osaka City University Medical Sch.,Osaka, Japan. Japanese Journal of Clinical Chemistry Vol. 21, No. 4, pp. 231-244 1992.
ISSN: 0370-5633. CODEN: RIKAAN
Pub. Country: Japan. Language: Japanese. Summary Language: English; Japanese.
Entered STN: 930307. Last Updated on STN: 930307

AB The aim of this study is to define the role of human C-reactive protein (CRP) in lipid metabolism, especially in relation to the binding capacity of CRP with serum lipoproteins (LP) by using gel filtration. The uptake of the CRP-LP complex through macrophage was observed by means of a light microscope. Column chromatography of hyperlipidemic human serum with or without the addition of CRP revealed that a complex was formed between CRP and LP, when the serum contained more than 180 mg/dl of total cholesterol and also with more than 110 mg/dl of triglyceride. CRP binds sufficiently with LP if the column is passed through with serum in which the titer of total cholesterol is around 250-299 mg/dl. Very low density lipoproteins (VLDL) were found to bind with CRP in the hyperlipidemic serum. Binding of CRP with LP was calcium ion-dependent and was inhibited by ligands such as phosphorylcholine or 6-amino-n-caproic acid. The CRP-LP complex was ingested by macrophage.

L13 ANSWER 6 OF 6 MEDLINE on STN

81239642. PubMed ID: 6788770. A novel phosphorylcholine-binding protein from rat serum and its effect on heparin-lipoprotein complex formation in the presence of calcium. Nagpurkar A; Mookerjea S. The Journal of biological chemistry, (1981 Jul 25) Vol. 256, No. 14, pp. 7440-6. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB An adsorbent was synthesized by attaching 4-aminophenylphosphorylcholine to cyanogen bromide-activated Sepharose. A phosphorylcholine (P-choline)-binding protein from rat serum was adsorbed on this affinity column which was eluted by 4 mM P-choline. The protein separated into two bands of Mr = 47,000 and 24,000 on sodium dodecyl sulfate-polyacrylamide gradient gels and contained 18% carbohydrate. A serum protein factor, precipitable by 30-50% (NH4)2SO4, was previously shown to inhibit

Ca2+-heparin-rat serum very low density lipoprotein (VLDL) precipitation reaction, whereas P-choline counteracted the action of this protein (Mookerjea, S. (1978) Can. J. Biochem. 56, 746-752). It is now demonstrated that purified P-choline-binding protein prevents Ca2+-heparin-chylomicron or VLDL complex formation and P-choline reverses the effect of this protein. Antibody to P-choline-binding protein raised in rabbits produced a single precipitin line against the pure antigen. The antiserum, however, did not react against rat serum chylomicron, VLDL, low density lipoproteins, or high density lipoprotein. Human serum appears to lack P-choline-binding protein, since (a) the affinity column did not adsorb any such protein, (b) P-choline had no effect on the Ca2+-heparin-serum lipoprotein precipitation reaction, and (c) an immunodiffusion test against the antiserum was negative. However, when P-choline-binding protein was added to human serum, the lipoprotein precipitation was inhibited, and P-choline counteracted the effect of this protein. Preliminary experiments suggested a stoichiometric interaction between P-choline-binding protein and VLDL. Hydrophilic P-choline groups exposed on the surface of VLDL may possibly interact with the P-choline-binding protein and thereby affect the precipitation of lipoproteins by heparin and Ca2+.

=> s monitoring disseminated intravascular coagulation
L14 1 MONITORING DISSEMINATED INTRAVASCULAR COAGULATION

=> d l14 cbib abs

L14 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
1992:78173 Document No.: PREV199293046628; BA93:46628. COAGULATION DISORDERS DUE TO MASSIVE BLOOD TRANSFUSION IN PERIPARTAL HYSTERECTOMY. REDDY S V G [Reprint author]; LINGAM S. DEP ANAESTHESIA, COLL MED, PO BOX 32485, AL-KHOD, MUSCAT, SULTANATE OF OMAN. International Journal of Feto-Maternal Medicine, (1991) Vol. 4, No. 1, pp. 27-30.
ISSN: 0933-0445. Language: ENGLISH.
AB The coagulation disorders associated with massive blood transfusion in 17 obstetric patients undergoing peripartal hysterectomy were studied. The indications for the procedure were postpartum hemorrhage, acute abruptio placentae, placenta acreta and ruptured uterus. The average amount of blood transfused in the intra-operative period was 5.3 1. Ringer's lactate was the only crystalloid used. Thrombocytopenia was the severest of the coagulation defects. The usage of blood products was limited to abnormal coagulation studies associated with obvious signs of bleeding. Two patients who had severe abruptio placentae died in the post-operative period of DIC. Close monitoring of the blood chemistry and regular screening of coagulation factors is required till the massively transfused obstetric patient is hemodynamically stabilized.

=> s monitoring
L15 1094150 MONITORING

=> s l15 and complex formation
L16 1273 L15 AND COMPLEX FORMATION

=> s l16 and coagulation
L17 40 L16 AND COAGULATION

=> s l17 and "C reactive protein"
L18 1 L17 AND "C REACTIVE PROTEIN"

=> d l18 cbib abs

L18 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:301143 Document No.: PREV200100301143. In vitro binding of C-

reactive protein to very low and intermediate density lipoproteins in a calcium-dependent complex. Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L. [Reprint author]. Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB The presence of a biphasic aPTT waveform has been correlated to negative outcome for patients in the intensive care setting (Downey C, et al. Early Identification and Prognostic Implications in Disseminated Intravascular Coagulation through Transmittance Waveform Analysis. Thromb Haemost 1998; 80: 65-9). It has recently been discovered that the mechanism underlying the atypical biphasic aPTT waveform is the result of the formation of a complex between C-reactive protein (CRP) with very low and intermediate density lipoprotein (VLDL or IDL) in a divalent cation-dependent reaction. The complex has been termed LC-CRP for "Lipoprotein-Complexed C-Reactive Protein". Initially, it was demonstrated that CRP isolated from patient plasma formed the complex when added to normal plasma. A possible mechanism for complex formation could be the result of qualitative differences in either CRP or the lipoproteins. To address this, recombinant CRP (rCRP) was combined in vitro with various lipoprotein subfractions. Free and bound CRP were quantified by ELISA. Results showed that rCRP was indistinguishable from patient CRP and that complex formation was specific for the VLDL/IDL subfraction. Recombinant CRP did not form a complex with LDL or HDL. Binding was inhibited by 1 mM phosphorylcholine, suggesting the involvement of the phosphatidylcholine lipid headgroups during complex formation. While these results may suggest that qualitative differences in CRP do not account for LC-CRP formation, they also enable the development of a quantitative assay for LC-CRP. An LC-CRP calibrator was constructed by mixing VLDL (0.24 mg/mL cholesterol) and CRP (200 mug/mL) in lipoprotein deficient plasma. By measuring the initial absorbance rate, dilutions in a plasma base provided a linear dose-response curve. As a synthetic VLDL substitute, artificial vesicles prepared from mixtures of phosphatidylcholine and lysophosphatidylcholine bound moderately to CRP. Development of a quantitative assay for LC-CRP that is fully automated and adaptable to multiple instrument platforms would allow direct measurement of the complex and has the potential to aid in therapeutic intervention and monitoring in this critically ill patient population.

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=> s l17 and VLDL
L19             1 L17 AND VLDL

=> d l19 cbib abs
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L19 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:301143 Document No.: PREV200100301143. In vitro binding of C-reactive protein to very low and intermediate density lipoproteins in a calcium-dependent complex. Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L. [Reprint author]. Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print. Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB The presence of a biphasic aPTT waveform has been correlated to negative outcome for patients in the intensive care setting (Downey C, et al.

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=> s l17 and IDL
L20          1 L17 AND IDL

=> d l20

L20  ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2006 The Thomson Corporation  on STN
AN   2001:301143  BIOSIS
DN   PREV200100301143
TI   In vitro binding of C-reactive protein to very low and intermediate
     density lipoproteins in a calcium-dependent complex.
AU   Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint
     author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L.
     [Reprint author]
CS   Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC,
     USA
SO   Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print.
     Meeting Info.: 42nd Annual Meeting of the American Society of Hematology.
     San Francisco, California, USA. December 01-05, 2000. American Society of
     Hematology.
     CODEN: BLOOAW. ISSN: 0006-4971.
DT   Conference; (Meeting)
     Conference; Abstract; (Meeting Abstract)
LA   English
ED   Entered STN: 27 Jun 2001
     Last Updated on STN: 19 Feb 2002


=> s l17 and chylomicron
L21          0 L17 AND CHYLOMICRON

=> s l17 and disseminated intravascular coagulation
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L22 1 L17 AND DISSEMINATED INTRAVASCULAR COAGULATION

=> d l22 cbib abs

L22 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:301143 Document No.: PREV200100301143. In vitro binding of C-reactive protein to very low and intermediate density lipoproteins in a calcium-dependent complex. Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L. [Reprint author]. Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print.
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CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

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=> s (fisher t?/au or downey c?/au or nesheim m?/au or samis j?/au or tejidor l?/au or walker j?/au)
L23 27786 (FISHER T?/AU OR DOWNEY C?/AU OR NESHEIM M?/AU OR SAMIS J?/AU OR TEJIDOR L?/AU OR WALKER J?/AU)

=> s l23 and monitoring complex formation
L24 0 L23 AND MONITORING COMPLEX FORMATION

=> s l23 and monitoring
L25 323 L23 AND MONITORING

=> s l25 hemostatic dysfunction
MISSING OPERATOR L25 HEMOSTATIC
The search profile that was entered contains terms or

nested terms that are not separated by a logical operator.

=> s l25 and hemostatic dysfunction
L26 2 L25 AND HEMOSTATIC DYSFUNCTION

=> dup remove l26
PROCESSING COMPLETED FOR L26
L27 2 DUP REMOVE L26 (0 DUPLICATES REMOVED)

=> d l27 1-2 cbib abs

L27 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2006 ACS on STN
2003:697159 Document No. 139:194022 Method for diagnosing and monitoring hemostatic dysfunction, severe infection and systematic inflammatory response syndrome. Toh, Cheng Hock; Tejidor, Liliana; Neisheim, Mike; Jones, Gregory (Biomerieux, Inc., USA). PCT Int. Appl. WO 2003073099 A1 20030904, 62 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US5980 20030227. PRIORITY: US 2002-359932P 20020227; US 2002-363073P 20020311; US 2002-396392P 20020717; US 2002-404652P 20020820.
AB A method for diagnosing and monitoring subjects for hemostatic dysfunction, severe infection and systematic inflammatory response syndrome is provided whereby lipoproteins are examined for abnormalities, particularly for prothrombinase enhancement, through quant. and qual. anal.

L27 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:305605 Document No.: PREV200100305605. The value of the APTT waveform in predicting mortality and haemostatic dysfunction. Toh, Cheng-Hock [Reprint author]; Downey, Colin [Reprint author]; Wenstone, Richard [Reprint author]; Paton, Ray [Reprint author]; Ticknor, Larry. Haematology, Anaesthesia and Computer Science, University of Liverpool, Liverpool, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 51a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.
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1988 score). However, all deaths in this group had evidence of haemostatic dysfunction with elevated markers of thrombin generation. Conclusion: The APTT waveform can identify ICU patients at risk of an adverse outcome both on and during admission. It has the potential too of shedding light on the pathophysiological interactions between coagulation, inflammation and the lipid response.

=> s l25 and
MISSING TERM AFTER L25 AND
Operators must be followed by a search term, L-number, or query name.

=> s l25 and c reactive protein
L28 5 L25 AND C REACTIVE PROTEIN

=> dup remove l28
PROCESSING COMPLETED FOR L28
L29 5 DUP REMOVE L28 (0 DUPLICATES REMOVED)

=> d l29 1-5 cbib abs

L29 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
2003:697159 Document No. 139:194022 Method for diagnosing and monitoring hemostatic dysfunction, severe infection and systematic inflammatory response syndrome. Toh, Cheng Hock; Tejidor, Liliana ; Neisheim, Mike; Jones, Gregory (Biomerieux, Inc., USA). PCT Int. Appl. WO 2003073099 A1 20030904, 62 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US5980 20030227. PRIORITY: US 2002-359932P 20020227; US 2002-363073P 20020311; US 2002-396392P 20020717; US 2002-404652P 20020820.
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L29 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
2003:435205 Document No. 139:19321 Method for predicting an increased likelihood of antiphospholipid syndrome in a patient using phospholipids and waveform analysis. Ortel, Thomas L.; Su, Zuowei; Braun, Paul J.; Tejidor, Liliana (USA). U.S. Pat. Appl. Publ. US 2003104493 A1 20030605, 47 pp. (English). CODEN: USXXCO. APPLICATION: US 2002-185186 20020628. PRIORITY: US 2001-302261P 20010629; US 2001-318755P 20010911.
AB A method for predicting that an individual has antiphospholipid syndrome or an increased likelihood of having antiphospholipid syndrome, includes: (a) providing a test sample from an individual; (b) combining the test sample with phospholipids; (c) directing a light beam at the test sample and monitoring light scattering or transmittance over time so as to provide a time-dependent measurement profile; (d) determining if a value or a slope at or over a particular time in the time-dependent measurement profile is beyond a corresponding predetd. value or slope threshold; and if the value or slope in the time-dependent measurement profile is beyond the predetd. threshold, then determining that the individual has antiphospholipid syndrome or an increased risk of antiphospholipid syndrome. The phospholipids can be provided as part of a coagulation reagent, or as part of a reagent where coagulation is not activated. Confirmatory assays for particular antibodies to phospholipid binding

proteins can be performed.

L29 ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:301143 Document No.: PREV200100301143. In vitro binding of C-reactive protein to very low and intermediate density lipoproteins in a calcium-dependent complex. Perez, U. [Reprint author]; Hoke, R. [Reprint author]; Doobay, H. [Reprint author]; Fischer, T. [Reprint author]; Samis, J.; Nesheim, M.; Tejidor, L. [Reprint author]. Hemostasis Reagent Development, Organon Teknika Corporation, Durham, NC, USA. Blood, (November 16, 2000) Vol. 96, No. 11 Part 2, pp. 76b. print.
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L29 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
2001:311730 Document No.: PREV200100311730. The biphasic waveform in the MDA system coagulation analyzer is due to the Ca++-induced formation and precipitation of a complex of very low density lipoprotein and C -reactive protein. Nesheim, Michael E.
[Reprint author]; Samis, John [Reprint author]; Walker, John [Reprint author]; Fischer, Tim; Tejidor, Liliana; Houdijk, Wim; Giles, Alan; Becker, Lev [Reprint author]; Koschinsky, Marlys [Reprint author]; Downey, Colin; Toh, Cheng Hock.
Biochemistry, Queen's University, Kingston, ON, Canada. Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 50a-51a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971. Language: English.

AB Downey, et al. recently showed that decreased light transmission prior to clot formation in the APPT assay using the Organon Teknika MDA-180 correlated with negative outcome for patients in intensive care (Thromb. and Haemost. 80, 65-9, 1998). The phenomenon was designated the "biphasic waveform" (BWF). In order to identify and characterize the molecular basis for the BWF, an assay based on the turbidity increase which occurs when mixtures of normal and patient plasmas are recalcified in the presence of a thrombin inhibitor was devised. The increase in turbidity was due to precipitate formation in positive samples. The precipitate was isolated from a pool of eight patient plasmas. Lipid and protein analysis showed that the precipitate contained components typical of very low density lipoprotein (VLDL) and C-reactive protein (CRP). Fractionation of the redissolved precipitate by ion exchange resolved the VLDL components from the CRP, neither of which alone would form a precipitate when recalcified, although a mixture of them would. Normal plasma supplemented with CRP formed a precipitate when recalcified. Further work showed that CRP readily forms a Ca++-dependent precipitate with purified VLDL and intermediate density lipoprotein (IDL), but not with low or high density lipoproteins. The interaction between normal VLDL and recombinant CRP exhibits a dissociation constant of 340nM (expressed relative to the CRP concentration) and VLDL at 1mM cholesterol binds 178mug CRP/ml. Binding is half maximal at 5.0mM Ca++. Fifteen patient plasmas with a BWF were analyzed with respect to the magnitude of the turbidity change upon reclacification and the concentrations of VLDL and CRP. The VLDL cholesterol levels ranged from 0.095 to 1.32mM, and the CRP levels were 77 to 398 mug/ml. The turbidity change correlated linearly with the VLDL level, but not with the level of CRP, which was

always in excess. We conclude that the BWF is due to formation of a very high molecular weight, Ca++-dependent complex between C-reactive protein, VLDL and possibly IDL.

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---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	186.85	187.06

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-7.50	-7.50

STN INTERNATIONAL LOGOFF AT 10:52:30 ON 10 OCT 2006